Manuscript Title: The effect of post-match alcohol ingestion on recovery from competitive Rugby League matches.

Running Head: Alcohol and Rugby League recovery.

**ABSTRACT**

This study investigated the effects of alcohol ingestion on lower body strength and power, and physiological and cognitive recovery following competitive Rugby League matches. Nine male Rugby players participated in two matches, followed by one of two randomized interventions; a control or alcohol ingestion session. Four hours post-match, participants consumed either beverages containing a total of 1g of ethanol per kg bodyweight (vodka and orange juice; ALC) or a caloric and taste matched non-alcoholic beverage (orange juice; CONT). Pre, post, 2 h post and 16 h post match measures of countermovement jump (CMJ), maximal voluntary contraction (MVC), voluntary activation (VA), damage and stress markers of creatine kinase (CK), C-reactive protein (CRP), cortisol, and testosterone analysed from venous blood collection, and cognitive function (modified Stroop test) were determined. Alcohol resulted in large effects for decreased CMJ height(-2.35 ± 8.14 and -10.53 ± 8.36 % decrement for CONT and ALC respectively; P=0.15, d=1.40), without changes in MVC (P=0.52, d=0.70) or VA (P=0.15, d=0.69). Furthermore, alcohol resulted in a significant slowing of total time in a cognitive test (P=0.04, d=1.59), whilst exhibiting large effects for detriments in congruent reaction time (P=0.19, d=1.73). Despite large effects for increased cortisol following alcohol ingestion during recovery (P=0.28, d=1.44), post-match alcohol consumption did not unduly affect testosterone (P-0.96, d=0.10), CK (P=0.66, d=0.70) or CRP (P=0.75, d=0.60). It appears alcohol consumption during the evening following competitive rugby matches may have some detrimental effects on peak power and cognitive recovery the morning following a Rugby League match. Accordingly, practitioners should be aware of the potential associated detrimental effects of alcohol consumption on recovery and provide alcohol allowance and awareness to athletes at post-match functions.

**Key words:** Team sport · Performance · Intermittent Sprint · Recovery

**INTRODUCTION**

Engagement in alcohol consumption is a common post-exercise practice for many athletes, particularly within the Rugby League community.21,23 The detrimental effects of pre-exercise alcohol consumption on physiological and performance outcomes are well reported,27 though considerably less is known about recovery following post-exercise alcohol consumption.1 Rugby League places much emphasis upon performance enhancement and recovery of athletes.10 Whilst alcohol ingestion following a match may be popular, it has the potential of slowing the rate of recovery due to its depressant nature on the circulatory and neuromuscular systems.26 Further, acute alcohol-induced perturbations of blood haemostasis,8 as well as chronic alcohol-induced glycolytic metabolism,16 neurogenic damage,16 and androgen receptor inhibition,34 may also suggest alcohol to have negative effects following exercise. However, to date few studies report the effects of alcohol on post-exercise recovery and no studies report the effects of alcohol consumption following team-sport competition. Such aforementioned ergolytic effects may impair subsequent recovery following matches thus slowing preparation for ensuing training sessions.

Alcohol consumption has been reported as disruptive to task performance through reduced reaction time, concentration, and memory loss.11 In Rugby League, such reduction in cognitive functioning, including decision making and reaction times may prove detrimental to athlete training development.9 Moreover, the negative effects of chronic alcohol ingestion are well documented in relation to a weakening of left ventricular contraction.22 Further, alcohol consumption may also result in blood lipid profile disruption, as well as, neutrophil, leukocyte, monocyte inhibition22 and increased hypohydration due to promotion of urine loss through inhibition of anti-diuretic hormone.17 Accordingly, whilst the acute and chronic consumption of alcohol is not physiologically advantageous for exercise, the effects on recovery following exercise are less well documented.1

In regard to recovery, the limited research suggests alcohol ingestion decreases peak concentric and eccentric voluntary torque 36 and 60h after 300 eccentric contractions of the quadriceps.1 As such, this reduction in skeletal muscle force following alcohol ingestion over the first 36h is likely to result in delayed recovery of performance.1 However, Barnes et al.1 reported that when exercise induced muscle damage is not present, the post-exercise ingestion of alcohol did not significantly affect maximal voluntary torque. Barnes, Mündel and Stannard2 further suggest that when exercise induced muscle damage is present; there is a greater decline in eccentric muscle recovery up to 60h post-exercise following alcohol consumption. Accordingly, despite limited evidence, it seems alcohol consumption may have a negative effect on post-exercise skeletal force production resulting in reduced recovery of voluntary force.

Despite these aforementioned findings, the majority of athletes refrain from alcohol consumption immediately prior to exercise on the day of a sporting event.24 However, many athletes may instead consume alcohol the night following competition, and are often then required to attend training and recovery session the following morning.24 Currently few studies investigate the effects of alcohol on recovery following the dynamic and multi-faceted requirements of field-based team sports. Accordingly, the aim of the current study was to investigate the effect of alcohol consumption on physiological and neuromuscular recovery the morning following a competitive rugby league match. It was hypothesized that alcohol ingestion would impede post-match recovery, specifically regarding an exacerbated physiological state and reduced neuromuscular function.

**METHODS**

*Experimental Approach to the Problem*

Following familiarisation, participants completed two testing conditions, 2-weeks apart, in a randomized fashion involving a control (CONT) and alcohol consumption (ALC) condition. Four hours following a competitive Rugby League match, subjects consumed either alcoholic or non-alcoholic beverages and remained overnight in the Laboratory until testing the following morning. Accordingly, this study simulated an ecologically valid sporting environment where players may consume alcohol the evening following a match, but are still required to attend strength, recovery or match analysis sessions the following morning. Venous blood samples, cognitive function and muscular performance were measured in the morning prior to the match (08:00), immediately post-match (occurring at standardised times within 5 – 10 min), 2h post-match, and 16h post-match the next morning (08:00). Competitive matches consisted of an official 80-min amateur rugby league match, commencing at 15:00 pm on each occasion. Participants were permitted 2h free-time following post-match testing to prepare for the night in the laboratory, in which subjects were required to maintain food/fluid diaries to replicate consumption and timing to standardize between conditions. The subjects returned at 18:00 for 2h post-match measures, followed by consumption of a standardized meal and then the respective interventions. Subjects were instructed to abstain from alcohol, caffeine and intense exercise 48h prior to initial testing. All fluid, food, physical activity, and sleep were standardized from 24h prior to initial testing and throughout testing using appropriate diaries or provision of food/fluid. These procedures were replicated during each session, including the inspection of diaries by the research team for subject compliance.

*Subjects*

Twelve well-trained, amateur, male Rugby League players volunteered to participate in this study. Due to injury or non-selection, final data sets involve 9 subjects with mean ± SD age 19.9±1.7y, body mass 84.1±11.2kg and stature 179.3±5.2cm. All subjects were of legal drinking age, physically trained and had at least 5 years prior rugby league experience at regional and amateur competitive level; participating in 2 x rugby-specific training sessions and 1-competitive match/week. All subjects were also associated as binge drinkers, which involved subjects consuming five or more drinks in a row at least once in the 2 weeks preceding the investigation. It should be further noted, that the legal age for alcohol consumption in Australia is 18 years and as such, all subjects were accustomed with drinking alcoholic beverages. All players were fully informed of the experimental procedures prior to providing written and verbal Informed Consent and Ethics were approved by the Institutional Ethics in Human Research Committee.

*Procedures*

*Rugby League Match*

Prior to the start of the match, players performed a match-specific warm up consisting of aerobic exercise, skill-based activities and stretching. Matches used throughout the study were of a standard equivalent to a level which is three grades below the National Rugby League competition, and chosen for mid-season rounds, home matches against opposition teams of similar abilities and competition standings (4th and 6th). During the match, players wore a Global Positioning Satellite device (SPI elite, GPSports Systems, Canberra, Australia) harnessed between the scapula to record distance and velocity of movement (1Hz). Data for distance and mean speeds collected during each match were reported in pre-defined categories; low-intensity activity (<14.5km.h-1), high-intensity running (14.5 - 20km.h-1) and very-high intensity running (>20km.h-1).7 Mean speed was calculated based on distance covered divided by the time spent in play (m.min-1). Previous research indicates that the technical error of measurement of these devices is <5% for total distance measured.7 Further, rate of perceived exertion (RPE)3 was measured 15-min following the conclusion of the match.

Matches were filmed to measure the number of collisions using a video camcorder (Canon MV920, Canon, Sydney, Australia) positioned at ground level on the half-way line of the field. Notational analysis was manually coded by a trained operator from video replays. Data were coded for number of tackles and hit ups to give a combined total of physical contacts.

*Intervention*

At 20:00, 4h following the match, subjects consumed a standardized volume and type of meal, consisting of lean chicken and vegetable kebabs, low-fat sausages, bread and mixed-salad (Energy: 30.5kj.kg-1; carbohydrate: 3.0g.kg-1; protein: 0.9g.kg-1). At 20:30, as per Barnes et al.,1 subjects began drinking a beverage containing either 1 g of alcohol per kg of body weight as vodka (37.5% alcohol/volume; Smirnoff, Australia) in orange juice (Westcliff, Minchinbury, NSW, Australia) or a control beverage of orange juice alone kept at a temperature of 4.5°C. Equivalent to 7.7±0.9 (2.77ml.kg-1) standard drinks (30ml), the mean volume of vodka consumed per subject was 233±29ml. In order to balance total energy value (3130±391kJ) and fluid volume (1788±223ml) between trials, subjects consumed a greater volume of orange juice in the CONT trial, while in ALC subjects consumed an additional volume of water (933±116ml) along with the alcoholic beverage. An equal volume of beverage was consumed every 20-min over a total time of 150-min, equating to a consumption of 11.9±1.5ml.min-1. Following consumption, participants prepared and then retired to self-provided bedding at 23:30. Subjects then slept in the darkened laboratory until the following morning at 07:00 for testing at 08:00.

*Measures*

*Muscular and Neuromuscular Performance*

Pre, post, 2h post and 16h post-match, participants completed a repeated counter-movement jump (CMJ) test to measure lower-body power. Prior to each CMJ (apart from post-match), participants performed a 5min warm up on a cycle ergometer (Ergomedic 828E Monark, Sweden) at 80rpm with 2kp resistance. Participants were required to perform 10-maximal, repeated CMJ’s using a force transducer (BMS encoder, Fitness Technologies, Australia) attached to a dowel rod across the shoulders and performed repeated jumps to determine peak and mean distance of the 10 jumps. Previous research indicates that the typical error of displacement using this measurement is 0.023 m.6

To determine changes in muscle contractile function, maximal voluntary contraction (MVC), voluntary activation (VA) and evoked muscle twitch contractile properties were assessed using an isokinetic dynamometer (Kincom, Model 125, Chattanooga Group Inc, Hixon, USA). Subjects were seated and strapped to the dynamometer across the chest, hips and near the ankle (1cm above the lateral malleolus) on the right leg, with arms crossed to isolate movement of the quadriceps. Participants’ hips and knee position were 90° flexion (0° represents full extension) and kept consistent between sessions using a manual goniometer. Muscle activation was achieved using a double felt-tip electrode (Bipolar felt-tip electrode, Cardinal Health, Madison, USA) placed over the femoral nerve on the anterior thigh 1.5cm below the inguinal fold. The electrical stimulus was delivered via a stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, England) linked to a BNC2100 terminal block and connected to a signal acquisition system (PXI1024; National Instruments, Austin, USA) which sampled all data at 2000Hz. Electrical stimulus was delivered using a single square-wave pulse with a width of 200µs, which was driven using customized software (v8.0, LabView; National Instruments).

Initially the current was applied in incremental steps until a plateau in peak twitch torque, and was then further increased by 10% to ensure supramaximal stimulation. Resting evoked twitches preceded the MVCs and included six pulses separated by 20s, delivered to the femoral nerve with the muscle at rest. Before the MVC protocol, isometric contractions were completed at 60%, 80% and 100% of maximal effort. The MVC protocol consisted of 15-maximal isometric contractions of the right knee extensor muscles30, maintaining maximal force for 3s, before a 6s recovery. The initial and final 5 contractions were superimposed with an electrical stimulus when peak torque was achieved (approximately 1s after initiation of the contraction) and a potentiated twitch was delivered immediately after the contraction was completed when the muscle was at complete rest.

Peak MVC was determined as the mean torque value produced during the MVC in the 50ms prior to the delivery of the stimulus. Voluntary activation (VA) levels were calculated using the twitch interpolation technique.30 Twitch contractile properties were analysed using mean torque-time curves from the potentiated evoked twitch contractions. Specific twitch properties which were extrapolated from the twitch contractile properties data included; peak potentiated twitch torque, time to peak torque and rate of torque development (MatLabTM Software;R2009b 7.9.0.529, The Mathworks Inc, USA).

*Physiological Measures*

Nude body mass was recorded pre, post, 2h post and 16h post match (BE-150K A1 Scales, A&D, Mercury PTY LTD, Thebarton, SA, Australia). Mid-stream urine samples were also collected for measurement of urine specific gravity (Pocket Refractometer, Atago, Japan), while throughout the night, subjects were required to urinate in a collection jar, as needed, to record urine output volume and weight.

Venous blood was collected at each time point and analysed for markers of stress, muscle damage and inflammation using an evacuated venipuncture system and serum separator tubes (Monovette, Sarstedt, Numbrecht, Germany). All samples reached room temperature, before centrifuged for 10min at 4000rpm; serum was removed and stored at -20ºC until analysis for Creatine Kinase (CK), C-reactive protein (CRP), testosterone, and cortisol. CK and CRP were analysed according to manufacturer’s instructions (Dimension Xpand spectrophotometer, Dade-Bearing, USA), with intra-assay CV’s of 0.61-0.64%, respectively. CK concentrations were determined using an enzymatic method and bichromatic rate technique.20 CRP samples were manually diluted according to manufacturer’s instructions and analysed with the particle enhanced turbidimetric immunoassay technique (PETIA). cortisol and testosterone concentrations were determined by a competitive immunoassay technique using chemiluminescent technology.

*Cognitive Assessment*

Cognitive function was assessed via a modified version of the Stroop test.32 This test of cognitive function was a computer based program requiring subjects to react to repeated colour and word stimuli. The program analysed response time and accuracy for congruent and incongruent stimuli. Measures of cognitive function were recorded pre, post, 2h post, post intervention, and 16h post-match.

*Statistical Analyses*

Data are reported as mean±SD. A repeated measures two-way ANOVA (condition x time) was used to determine significant differences for time and time by treatment, within and/or between each condition, with significance at P<0.05, and differences considered as trends when 0.05>p<0.10. Given the lack of standardized work during the match, a one-way ANOVA was also used to determine significant differences in the percentage change of measures from 2h - 16 h post-match (i.e., 2h post until the next morning), as this is the duration of time in which the intervention was imposed. All data collected were analysed using SPSS version-14.0 (Chicago, USA). Further, effect sizes (ES; Cohen’s d) were calculated to determine the magnitude of effect of alcohol consumption on recovery. Effects sizes <0.40 were considered small, 0.40-0.70 moderate, and >0.80 large effect size.

**RESULTS**

*Match Responses*

Total, high intensity running and very high intensity running distances, total contacts and RPE for each condition are presented in Table 1. Mean playing time for ALC was 72.2±12.3 min and 75.4±6.2 min for CONT of the 80-min match. Total distances covered were significantly greater during CONT conditions (P=0.02;d=1.80); however, no significant differences were apparent for m.min-1, high intensity running or very high intensity running, total contacts, or RPE between conditions (P=0.10–0.90; d=0.10–0.60).

(Table 1 about here)

*Neuromuscular Performance*

Comparisons between conditions showed no significant differences for peak or mean maximal distance in CMJ regarding change over time (P=0.32-0.36; Table 2). Further, no significant differences in percentage change from 2h-16h post-match were observed between conditions for peak or mean maximal distance (P=0.15-0.73;Table 2). However, large ES were evident for an attenuated change in peak maximal distance (d=1.40) and mean maximal distance (d=1.09) with ALC the morning following the match.

Significant reductions in MVC over time were observed in both conditions between pre and all further time points (P=0.01; Table 2). However, no significant change in VA was evident over time in either condition (P=0.42; Table 2). Further, no significant differences were apparent between conditions for MVC (P=0.52; d=0.70) or VA (P=0.15; d=0.69), respectively. Percent change from 2h-16h post-match for mean MVC (P=0.25;d=0.69) or VA (P=0.33;d=0.69), respectively were not significantly different between conditions.

 *Twitch contractile properties*

Analysis of peak potentiated twitch torque and rate of torque development indicated significant changes during ALC over time for post-match compared to pre-match (P=0.03-0.04). Similarly, a significantly greater peak potentiated twitch torque in ALC compared to CONT was present (P=0.008). However, there was no significant difference in % change between conditions 2h -16h post-match for peak potentiated twitch torque or rate of torque development (P=0.21-0.82; d=0.20; Table 2). No significant differences in time to peak torque were present over time (P=0.19) in either condition. Furthermore, there was no significant difference between conditions ALC and CONT for time to peak torque (P=0.28). However, there was a significantly greater % reduction in time to peak torque for CONT from 2h-16h post match (P=0.04; d=1.60; Table 2).

(Table 2 about here)

*Biochemical Variables and Nude Body Mass*

Biochemical responses of stress, damage and inflammation to match-play and subsequent interventions are presented in Figure 1. CK concentrations were significantly increased over time (P=0.001), whilst CRP indicated no significant difference over time (P=0.18). No significant differences were apparent between conditions for CK or CRP (P=0.66; d=0.70; P=0.08;d=0.60). Furthermore, CK and CRP demonstrated no significant differences between conditions in % change from 2 -16 h post match (P=0.32-0.43). Testosterone was reduced 2h post match (P=0.001), followed by a significant increase to 16h post-match (P=0.001). Cortisol demonstrated significant decreases after the match (P=0.001) followed by a significant increase 16h post-match (P=0.001). However, there were no differences in testosterone or cortisol responses between conditions (P=0.96, d=0.10; P=0.28) or regarding % change from 2h-16 h post-match (P=0.49; P=0.07). However, large ES for % change from 2-16h post-match were observed for the increase in cortisol response for ALC (d=1.40).

Post-intervention urine output (ALC 1400±502ml; CONT 1075±400ml), change in nude mass (CONT -0.1±1.2; ALC-0.3±1.2kg), and urine specific gravity at pre, 2h post and 16h post (CONT 1.021±0.005, 1.029±0.004, 1.022±0.007; ALC 1.021±0.008, 1.022±0.008, 1.022±0.004 respectively) showed no significant differences between conditions (P=0.15; P=0.61; P=0.36 respectively) between conditions, although over-night urine output demonstrated large ES for increased total volume in ALC (d=1.09).

(Figure 1 about here)

*Cognitive and Perceptual Responses*

Comparisons between conditions show no significant differences for cognition test time, congruent reaction time or incongruent reaction time regarding change over time (P=0.09; P=0.16; P=0.20 respectively). Cognition test time significantly increased for ALC compared to CONT (P=0.04); however, congruent reaction time and incongruent reaction time demonstrated no significant difference between conditions (P=0.19; P=0.29 respectively; Table 2. Moreover, there was a significant increase in congruent reaction time for ALC compared with CONT (P=0.02) for % change from 2h-16 h post-match. Further, there were large ES for an increase in cognition test time, congruent reaction time and incongruent reaction time for ALC compared to CONT (d=1.59; d=1.73; d=0.80 respectively; Table 2).

**DISCUSSION**

The aim of this study was to examine the effects of post-match alcohol consumption on recovery following competitive rugby league matches. Results suggest that the ingestion of alcohol had no significant effect on skeletal muscle contractile force or activation, although large effects for a suppression of CMJ the next morning were present. Further, there were no significant differences in testosterone, CK or CRP measures due to alcohol ingestion. However, cortisol responses demonstrated large effects for lower values following ALC. Moreover, ALC appears to negatively influence recovery of cognitive function the morning following a competitive match. Accordingly, it appears that acute alcohol consumption of binge drinkers may have some detrimental effects on recovery the morning following a Rugby League match.

In regards to the recovery of performance following alcohol ingestion in the present study, no significant differences were present for CMJ. However, it seems that mean and peak power may be negatively affected by ALC, as large effects were present for %change (2–16h post-match) in reductions in peak and mean CMJ compared to CONT. To date, no literature reports the effect of ALC on recovery of CMJ. However, regarding alcohol ingestion 30-min prior to CMJ testing, Hebbelinck12 has reported a decrease in dynamic power of 5.8% following ingestion of 94% ethyl alcohol (0.6mL.kg-1). Further, such findings have also been related to slower 80-m sprint time following alcohol,12 suggesting total-body power output may be compromised by alcohol ingestion. Similarly, the current results indicate a large effect of a suppression of CMJ following post-match alcohol consumption. Even without alcohol consumption, elite rugby league matches are known to reduce peak power, as McLellan et al.,19 recently reported decreased CMJ peak force and peak rate of force development for up to 48h post-match. Force production responses in the current investigation appear inconsistent with previous similar investigations (i.e. Barnes et al., 2) in that CMJ did not significantly decrease between prior time points compared to the significant decrements in isometric, concentric, and eccentric torque production reported by Barnes et al.2 Accordingly, the observed decrease in CMJ 16h post-match may be due to the ALC intervention itself, rather than represent an interrupted recovery process. It should be noted that alcohol use in the chronic sense is a stressor in which the main influence comes from repeated use along with the inability to tolerate it; with tolerance dependent upon a variety of factors including drinking history, and dose.14 However, often team-sport athletes, particularly rugby league players, will engage in acute binge drinking acts following weekly competitive matches, as such the present study simulated such practices. Regardless, the current results should be taken in context of a dose of alcohol that may not exceed the regular drinking practices of the subject population.

MVC, VA and contractile properties did not differ between conditions over the duration of testing. Poulsen et al.25 have reported no detriment to MVC up to 48h following alcohol ingestion; however, these results were recorded in a rested state. Clarkson and Reichsman5 suggest a lack of reduction in muscle strength may relate to below optimal alcohol ingestion, referring to individual subject acute alcohol tolerance not sufficiently promoting adverse effects on excitation-contraction coupling. In the present study, there was no difference between conditions for twitch contractile property measures, suggesting no apparent effect on peripheral contractile mechanisms due to ALC. However, it is possible in the present study that the volume of alcohol was lower than that observed during normal social drinking for this population, as unfortunately no measure of blood alcohol content was recorded. Furthermore, due to the ingestion of the standardised meal in such close proximity to the alcohol intervention, the peak blood alcohol level may have been attenuated, further blunting the effect of alcohol on muscle function. Accordingly, such an alcoholic volume may not have affected central recruitment (VA), peripheral mechanisms (contractile properties) or resulting voluntary force (MVC). Moreover, eccentric loading during the Rugby League match may not have been sufficient to induce isolated exercise induced muscle damage as has previously been reported from 300 single-leg eccentric contractions.1 However the distance travelled during the match (6221±701.1m) would suggest a sufficient external load to be responsible for the suppression of MVC. Alternatively, no apparent difference in VA or twitch contractile properties suggests that alcohol had no interference with neural drive or muscle recruitment.2, 33 Consequently, while skeletal muscle function was not affected, subjects may have made a conscious decision to reduce jump height following ALC; possibly due to motivation effects resulting from prior intoxication or from the current ‘hang-over state’. Regardless, despite large effect sizes indicating suppressed total-body peak power, no significant effect of alcohol ingestion was noted for voluntary or evoked quadriceps force.

Although single-leg, muscle-specific damage may not have been present in this study, both CK and CRP showed post-match elevations, indicative of some skeletal muscle damage.20,19,31 Minett et al.20 suggest that elevations in CK are indicative of muscle degradation and contusion associated trauma in rugby, which may have been due to inter-athlete contact and/or eccentric loading. The current CK and CRP data is comparable to muscle damage and inflammation reported in other studies, typically inducing increased CK up to 120h post-match.19 Further, as there were no significant differences between conditions, the present dose of alcohol may have minimal effect on muscle damage or inflammation markers during post-match recovery. Similarly, testosterone responses during recovery did not indicate significant differences between conditions, which are comparable to research by Koziris et al.15,who reported no effect on testosterone following resistance exercise session both with and without ethanol ingestion. Such a finding differs to reports by Heikkonen et al.,13 who reported decreased testosterone following exhaustive ergometer exercise and ensuing ingestion of alcohol. One possible reason for the difference in findings is that the match was not of sufficient intensity to induce a significant increase in mean serum testosterone as observed by Heikkonen et al.13 Conversely, cortisol data indicated large effects in the ALC condition with greater % increase 2h-16h post-match compared to CONT. However, it is also important to note that cortisol levels at 2h post-match were lower in the ALC condition, whist returning to similar levels 16h post-match, possibly prompting the findings of a large increase. It is conceivable that such a response was due to discrepancies in match external loads, as demonstrated through the differences in total distance covered between matches. In contrast to these findings, Heikkonen et al.13 reported no change in serum cortisol concentration between alcohol (1.5g.kg-1) and non-alcohol conditions measured 3h following a 30-min cycle protocol. Heikkonen et al.13 suggest that the lack of significant change in cortisol was unexpected, although it was suggested that a greater increase in cortisol was observed in the ALC condition as alcohol may directly stimulate cortisol production of the adrenal glands.13 Furthermore, the present findings are analogous to Koziris et al.15 with no significant differences found in cortisol responses between exercise bouts due to ethanol ingestion. Koziris et al.15 suggest that the amount of alcohol consumed was not enough to acutely increase cortisol above the exercise only condition in their study; which may also represent the responses noted here. Although no significant differences were observed in the present study, large effects were present in the ALC condition for a reduction in cortisol 2-16h post-match, which may further suggest that damage did occur, as a typical biochemical response of rugby league due to heavy physical contact19 is evident with an increased level of cortisol (catabolic) responses.20

In regards to hydration status, alcohol is reported to negatively affect hydration through the inhibition of vasopressin secretion29 with the degree of dieresis proportional to the amount of alcohol consumed.29 In the present study, large effects for increased urine output in ALC suggest that alcohol ingestion during recovery may induce increased urinary output, and as such, may be indicative of reduced hydration state or the need to consume greater (non-alcoholic) fluid volumes. Such effects concur with Maughan et al.18 who report increased urine output and dehydration due to ingestion of drinks >4% alcohol. However, in the present study, there were no significant differences between conditions for urine specific gravity. Previously, Shirreffs and Maughan29 report that alcohol ingested whilst in a hypohydrated state blunts the diuretic effect of alcohol. Furthermore, that alcohol inhibits antidiuretic hormone and acts detrimentally upon urinary output during recovery.29 Following the present match, urine specific gravity values indicated that increased hypohydration occurred post match for both conditions. As the dehydrated state of participants was evident post-match, it remains unclear the full extent of alcohol regarding post-match hydration.

Apart from strength, power and physiological state, recovery of cognitive function following a match may be important for ensuing team-sport training sessions. Results from the present study corroborate previous findings that highlight that post-alcohol consumption (i.e. “hangover”) is detrimental to cognition, particularly with the observed significant negative differences for total Stroop test time.28 Alcohol is reported to negatively inhibit neural outcomes through disruptions in multiple neurotransmitter systems by altering inhibitory and excitatory neurotransmission33. Accordingly, alcohol ingestion during recovery may reduce cognitive functioning; particularly decision making speed and quality of responses to visual stimuli. In rugby league, a reduction in cognitive functioning such as decision making and reaction times may prove detrimental to athlete training progression; as successful kinaesthetic learning outcomes may be hindered.9

Before conclusion of the present findings, a brief dialogue concerning the limitations of the study is in order. Apart from the reduced subject number, the ecologically valid environment of the present study resulted in variance in match demands due to different opponents and workloads. Despite these limitations, attempts were made to ensure similar demands through randomization of conditions, scheduling of testing to coincide with home matches and oppositions of similar playing status. As a result, match outcomes were similar for both conditions with matches being won by 2 and 8 points respectively. Through analysis of global positioning system data for distance and speed, RPE and notational analysis of total physical contacts, it was evident that there was no significant variance between matches for high-intensity work. However, an exception was a larger total distance covered in CONT, which may indicate a larger external load. A further consideration is that glycogen storage and energy intake implications resulting from alcohol ingestion have been previously reported to displace carbohydrate intake from optimal recovery nutrition methods, indirectly resulting in impairments of carbohydrate and lipid metabolism.4 Specifically, these are highlighted not to be as a result of alterations in metabolism, but rather due to the consequences of the environment in which alcohol is typically consumed i.e., athletes in real life will not ingest sufficient carbohydrate required for adequate muscle recovery4. While muscle glycogen content was not measured, glycogen depletion was avoided during the current study due to a standardized diet which included sufficient ingestion of carbohydrate. Finally, due to the ecological setting of the study, a standardized exercise load was not enforced, potentially resulting in variability of responses and when combined with a small subject number, reduced statistical power of the study, which are also acknowledged as a limitation.

In conclusion, the present study sought to determine the effect of alcohol on post-match performance, physiological and perceptual recovery. Muscular performance responses to alcohol resulted in decreased CMJ, without changes in MVC, VA or twitch contractile properties. Furthermore, alcohol was observed to be detrimental to the speed and accuracy of cognitive response to visual stimuli. However, despite some alterations in cortisol responses, post-match alcohol consumption did not unduly affect CK or CRP markers of damage. Although some variability in match demands was observed, it seems that alcohol consumption can exhibit some negative effects on ensuing post-match recovery. Future research should further explore the effect of acute post-match alcohol consumption with varying levels of intoxication, as it appears that a lack of inherent intoxication could blunted the detrimental effects of alcohol towards recovery. Regardless, practitioners should be aware of such possible detrimental effects, and regulate the consumption of alcohol accordingly in light of the potential physiological, perceptual and cognitive consequences.

**PRACTICAL APPLICATIONS**

This investigation demonstrates that the ingestion of alcohol has a negative effect on post intermittent exercise performance resulting in detriments in muscle performance and cognition. Specifically, alcohol ingestion during recovery has shown a large effect of a suppression of CMJ following post-match alcohol consumption negative impact on CMJ and cognitive tasks of reaction time and word- color association tasks. Therefore, these detriments must be taken into consideration when planning subsequent training and competitive matches. Further, findings from this study suggest that recovery following rugby league matches appears to be impaired when alcohol consumption is present. Accordingly, alcohol should be avoided, or managed correctly, during recovery within 16 h following a competitive rugby league match regardless of the use of other recovery modalities. Furthermore, with regards to alterations in metabolism, alcohol may interfere with an athlete’s ability or interest to replenish carbohydrate levels to a sufficient level for glycogen resynthesis to occur. Thus, practitioners must be aware that when alcohol ingestion practices are planned post-match, due to certain social obligations, prior carbohydrate ingestion takes place.

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**Figure Captions**

**Figure 1:** Mean ± SD creatine kinase (CK), C-reactive protein (CRP), cortisol, and testosterone for control (CONT, n = 9), and alcohol (ALC, n = 9) for pre, post, 2h post and 16h post match measures and change (%) 2 h post to 16 h post match. ‡ Significant (P<0.05) difference over time compared to pre-match.† Large effect size (d>0.8) comparison.